ORIGINAL

Arizona Corporation Commission Docket E-00000C-11-0328

To: The Arizona Corporation Commission and Interested Parties

From: Smart Meter Research

You may be feeling healthy but your blood is telling a different story.

Dr Dietrich Klinghardt MD, PhD has found high levels of TGF Beta-1, MMP9, and copper in serum of patients he has treated and monitored for up to 10 years. The results are noticed once a smart meter has been installed.

Attached is Dr Klinghardt's Bio Data along with definitions of the inflammatory markers mentioned.

PubMed

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SpringerLink FULL-TEXT ARTICLE

Med Oncol. 2012 Dec;29(5):3394-9. doi: 10.1007/s12032-012-0283-z. Epub 2012 Jun 30.

Matrix metalloproteinase-9 is a prognostic marker for patients with cervical cancer.

Li Y, Wu T, Zhang B, Yao Y, Yin G.

Department of Gynaecology and Obstetrics, Tangdu Hospital, The Fourth Military Medical University, Xi'an, Shaanxi, China.

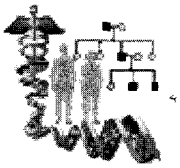
Abstract

Cervical cancer remains one of the most common malignancies in women. Previous study proved MMP-9 might be prognostic marker for multiple human malignancies. The present study was to investigate the protein expression of MMP-9 in cervical cancer and its association with clinicopathological characteristics as well as prognosis of patients. Cervical cancer specimens from 225 cases who had not received chemotherapy or radiotherapy prior to surgery were collected. Immunochemistry assays were utilized to investigate MMP-9 protein expression. Results showed that MMP-9 expression was increased in cervical cancer and associated with stromal invasion, FIGO stage, lymph node metastasis, and vascular invasion. Kaplan-Meier analysis showed that patients with cervical cancer of positive MMP-9 staining tend to have worse overall survival. In multivariate analysis stratified for known prognostic variables, MMP-9 was proved to be an independent prognostic factor. The present study confirmed that MMP-9 expression in cervical cancer was an independent prognostic factor of patients, which might be a potential diagnostic and even therapeutic target of cervical cancer.

PMID: 22752570 [PubMed - indexed for MEDLINE]

MeSH Terms, Substances

LinkOut - more resources



Genetics Home Reference
Your Guide to Understanding Genetic Conditions
http://ghr.nlm.nih.gov/ A service of the U.S. National Library of Medicine®

TGFB1

Reviewed April 2008

What is the official name of the *TGFB1* gene?

The official name of this gene is "transforming growth factor, beta 1."

TGFB1 is the gene's official symbol. The *TGFB1* gene is also known by other names, listed below.

What is the normal function of the *TGFB1* gene?

The *TGFB1* gene provides instructions for producing a protein called transforming growth factor beta-1 (TGFβ-1). The TGFβ-1 protein helps control the growth and division (proliferation) of cells, the process by which cells mature to carry out specific functions (differentiation), cell movement (motility), and the self-destruction of cells (apoptosis). The TGFβ-1 protein is found throughout the body and plays a role in development before birth, the formation of blood vessels, the regulation of muscle tissue and body fat development, wound healing, and immune system function. TGFβ-1 is particularly abundant in tissues that make up the skeleton, where it helps regulate bone growth, and in the intricate lattice that forms in the spaces between cells (the extracellular matrix). Within cells, this protein is turned off (inactive) until it receives a chemical signal to become active.

How are changes in the *TGFB1* gene related to health conditions?

Camurati-Engelmann disease - caused by mutations in the *TGFB1* gene

Approximately 10 mutations in the *TGFB1* gene have been found to cause Camurati-Engelmann disease. Most of the mutations change one protein building block (amino acid) in the TGFβ-1 protein. The most common mutation replaces the amino acid arginine with the amino acid cysteine at position 218 in the TGFβ-1 protein (written as Arg218Cys or R218C).

All mutations that cause Camurati-Engelmann disease result in a TGFβ-1 protein that is always turned on (active). The overactive protein likely disrupts the regulation of bone growth and impairs muscle and body fat development. A disruption in the regulation of TGFβ-1 activity can lead to increased bone density and other features of Camurati-Engelmann disease.

cancers - associated with the *TGFB1* gene

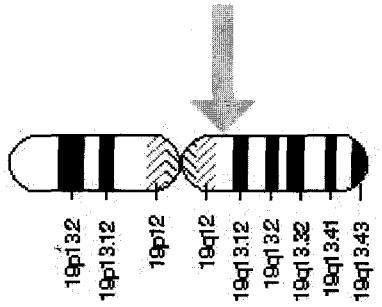
Some *TGFB1* gene mutations are acquired during a person's lifetime and are present only in certain cells. These changes are called somatic mutations and are not inherited. Somatic mutations in the *TGFB1* gene that cause alterations in the activity (expression) of the TGFβ-1 protein are associated with certain cancers. The altered protein expression may enhance several cancer-related events such as cell division (proliferation), cell motility, and the development of new blood vessels (angiogenesis) that nourish a growing tumor. The TGFβ-1 protein is abnormally active (overexpressed) in certain types of prostate cancers. Altered TGFβ-1 expression has also been found in breast, colon, lung, and bladder cancers.

A variation (polymorphism) in the *TGFB1* gene that changes a single amino acid in the TGFβ-1 protein is associated with prostate cancer. In people with this polymorphism, the amino acid leucine is replaced with the amino acid proline at position 10 in the TGFβ-1 protein. Although it has no apparent effect in healthy people or those with a condition caused by a different mutation in the *TGFB1* gene, this polymorphism is associated with accelerated disease progression and a poorer outcome in patients with prostate cancer.

Where is the *TGFB1* gene located?

Cytogenetic Location: 19q13.1

Molecular Location on chromosome 19: base pairs 41,836,811 to 41,859,830



The *TGFB1* gene is located on the long (q) arm of chromosome 19 at position 13.1.

More precisely, the *TGFB1* gene is located from base pair 41,836,811 to base pair 41,859,830 on chromosome 19.

See How do geneticists indicate the location of a gene? (http://ghr.nlm.nih.gov/handbook/howgeneswork/genelocation) in the Handbook.

Where can I find additional information about *TGFB1*?

You and your healthcare professional may find the following resources about *TGFB1* helpful.

- **Educational resources - Information pages**
  - Cancer Medicine (sixth edition, 2003): Transforming Growth Factor-β (TGF-β) (http://www.ncbi.nlm.nih.gov/books/NBK12565/)
  - Eurekah Bioscience Collection: TGFβ Signaling (http://www.ncbi.nlm.nih.gov/books/NBK6525/)
  - Molecular Cell Biology (fourth edition, 2000): TGFβ signaling pathway (http://www.ncbi.nlm.nih.gov/books/NBK21715/?rendertype=figure&id=A6758)
- Gene Reviews - Clinical summary (http://www.ncbi.nlm.nih.gov/books/NBK1156/)
- **Genetic Testing Registry - Repository of genetic test information**
  - GTR: Genetic tests for TGFB1 (http://www.ncbi.nlm.nih.gov/gtr/tests/?term=7040%5Bgeneid%5D)

You may also be interested in these resources, which are designed for genetics professionals and

researchers.

- PubMed - Recent literature (http://www.ncbi.nlm.nih.gov/pubmed?term=((TGFB1%5BTIAB%5D)%20OR%20(TGF%20beta-1%5BTIAB%5D))%20AND%20((Genes%5BMH%5D)%20OR%20(Genetic%20Phenomena%5BMH%5D))%20AND%20english%5Bla%5D%20AND%20human%5Bmh%5D%20AND%20%22last%201800%20days%22%5Bdp%5D)
- OMIM - Genetic disorder catalog (http://omim.org/entry/190180)
- **Research Resources - Tools for researchers**
  - Atlas of Genetics and Cytogenetics in Oncology and Haematology (http://atlasgeneticsoncology.org/Genes/TGFB1ID42534ch19q13.html)
  - Cancer Genetics Web (http://www.cancerindex.org/geneweb/TGFB1.htm)
  - Entrez Gene (http://www.ncbi.nlm.nih.gov/gene/7040)
  - GeneCards (http://www.genecards.org/cgi-bin/carddisp.pl?id_type=entrezgene&id=7040)
  - HUGO Gene Nomenclature Committee (http://www.genenames.org/data/hgnc_data.php?hgnc_id=11766)

What other names do people use for the *TGFB1* gene or gene products?

- CED
- diaphyseal dysplasia 1, progressive
- DPD1
- TGFB
- TGFB1_HUMAN
- TGFbeta
- TGF-beta-1
- TGF-beta 1 protein
- transforming growth factor beta 1
- transforming growth factor-beta 1
- transforming growth factor, beta 1 (Camurati-Engelmann disease)

See How are genetic conditions and genes named? (http://ghr.nlm.nih.gov/handbook/mutationsanddisorders/naming) in the Handbook.

What glossary definitions help with understanding *TGFB1*?

amino acid ; angiogenesis ; apoptosis ; autocrine ; bone density ; cancer ; cell ; cell division ; colon ; differentiation ; dysplasia ; extracellular ; extracellular matrix ; gene ; growth factor ; immune system ; leucine ; mutation ; paracrine ; polymorphism ; progression ; proliferation ; prostate ; protein ; tissue ; tumor

You may find definitions for these and many other terms in the Genetics Home Reference Glossary (http://ghr.nlm.nih.gov/glossary).

References

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- Wallace SE, Lachman RS, Mekikian PB, Bui KK, Wilcox WR. Marked phenotypic variability in progressive diaphyseal dysplasia (Camurati-Engelmann disease): report of a four-generation pedigree, identification of a mutation in TGFB1, and review. Am J Med Genet A. 2004 Sep 1;129A(3):235-47. (http://www.ncbi.nlm.nih.gov/pubmed/15326622?dopt=Abstract)

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PubMed

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Performing your original search, ***copper toxicity***, in PubMed will retrieve **7722** records.

J Am Coll Nutr. 2009 Jun;28(3):238-42.

The risks of copper toxicity contributing to cognitive decline in the aging population and to Alzheimer's disease.

Brewer GJ.

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Abstract

It is a pleasure and an honor to contribute a paper to a special issue of the Journal of the American College of Nutrition honoring Stanley Wallach and Pearl Small. In this brief review I advance the hypothesis that copper toxicity is the major cause of the epidemic of mild cognitive impairment and Alzheimer's disease engulfing our aging population. This epidemic is recent, exploding in the last 50-60 years. The disease was virtually unknown 100 years ago. And it involves only developed countries that use copper plumbing. Something in our environment associated with development is poisoning the minds of our aged. The epidemic is associated with the use of copper plumbing, and the taking of copper in multi-mineral supplements. Food copper (organic copper) is processed by the liver and is transported and sequestered in a safe manner. Inorganic copper, such as that in drinking water and copper supplements, largely bypasses the liver and enters the free copper pool of the blood directly. This copper is potentially toxic because it may penetrate the blood/brain barrier. I review a web of animal and human data that tightens the noose around the hypothesis that copper toxicity is causing the epidemic of Alzeimer's disease and loss of cognition in our aging population.

PMID: 20150596 [PubMed - indexed for MEDLINE]

Publication Types, MeSH Terms, Substances

LinkOut - more resources

DR DIETRICH KLINGHARDT MD PHD

Dr Dietrich Klinghardt Bio Data

Dr Klinghardt - Dr Dietrich Klinghardt MD, PhD, is Founder of the Klinghardt Academy (USA), the American Academy of Neural Therapy, Medical Director of the Institute of Neurobiology, and lead clinician at the Sophia Health Institute, located in Woodinville, Washington. He is also Founder and Chairman of the Institute for Neurobiology (Germany) and (Switzerland). Klinghardt Academy (USA) provides teachings to the English speaking world on biological interventions and Autonomic Response Testing assessment techniques.

Klinghardt has lectured at the universities of Illinois, Utah, Freiburg, Adelaide, Capital University (Washington DC) and others, and the medical schools of Geneva and Zurich. Between 1996-2005 he was Associate Professor at the Department of Applied Neurobiology at Capital University. He is regularly invited to teach workshops at the prestigious Medicine Week in Baden-Baden, Germany and the International Lyme and Associated Diseases (ILADS) conferences. Among his books is the groundbreaking Psychokinesiology A new Approach in Psychosomatic Medicine, on muscle feedback-guided psychotherapy. Many of his teachings, manuals, seminar DVD's and clinical tools are available through his website www.klinghardtacademy.com.

Klinghardt Treatments

Internationally known for his successful treatment of chronic pain and illness, Dr. Klinghardt combines non-surgical orthopaedic medicine with immunology, endocrinology, toxicology, neural therapy, hypnotherapy and energy psychology. His unique approach to diagnosing and treating diseases and disorders on both the physical and mental-emotional levels recognises that good health is dependent on:

A well-functioning autonomic nervous system
A healthy mind that creates a balanced emotional state
A supportive network of relationships within current and past generations of the family. His teaching includes explorations of the influence of family relationships on health, based on approaches developed by psychoanalyst Bert Hellinger

Klinghardt Studies

Klinghardt studied medicine (1969-1975) and psychology (1975-1979) in Freiburg, Germany, completing his PhD on the involvement of the autonomic nervous system in autoimmune disorders. Several publications followed. Early in his career, he became interested in the sequelae of chronic toxicity (especially lead, mercury, environmental pollutants and electromagnetic fields) for the course of illness. While working in India as a junior physician, he encountered Eastern concepts of disease etiology and blended them with his Western training. This laid the foundation for his 5-level system of Integrative Medicine.

Dr Klinghardt As Physician

After immigrating to the USA, he spent three years as a full-time emergency physician before becoming Medical Director of the Santa Fe Pain Centre. Increasingly aware of the limitations of conventional medicine when dealing with chronic conditions, he trained in Ericksonian hypnotherapy and began to include body-oriented psychotherapeutic and counselling approaches in his work, along with neural therapy, mesotherapy injection techniques and applied psychoneurobiology (psychokinesiology and mental field therapy).

Since the 1970s, Dr. Klinghardt has contributed significantly to the understanding of metal toxicity and its connection with chronic infections, illness and pain. He is considered an authority on this subject and has been instrumental in advancing various fields within biological medicine - non-invasive pain management, injection techniques for pain and orthopaedic dysfunction, anti-ageing medicine, toxicology, paediatrics (neuro-developmental disorders), energy psychology, biological dentistry, and others. He has also developed Autonomic Response Testing, a comprehensive diagnostic system that has helped many practitioners to become accomplished holistic physicians.

Klinghardt has lectured at the universities of Illinois, Utah, Freiburg, Adelaide, Capital University

(Washington DC) and others, and the medical schools of Geneva and Zurich. Between 1996-2005 he was Associate Professor at the Department of Applied Neurobiology at Capital University. He is regularly invited to teach workshops at the prestigious Medicine Week in Baden-Baden, Germany. Among his books is the groundbreaking Psychokinesiology A new Approach in Psychosomatic Medicine, on muscle feedback-guided psychotherapy.

Dietrich Klinghardt Work

In recognition of his work, Klinghardt received the Physician of the Year award from the Global Foundation of Integrative Medicine in May 2007, and the 2011 Physician of the Year Award for the International Academy of Biological Dentistry and Medicine.

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